

Ameliorative Effect of Glutathione Supplementation Against Imidacloprid Toxication In Japanese Quails

Eman G.E.Helal*, Samir A.M.Zahkook*, Nabil Fahmy**, Mohammed S.A.A Al-Shinnawy*** and Amira B.Abd-El-Ghany*

* Zoology Department, Faculty of Science (Girls), Al-Azhar University.

** Animal Production Department, Faculty of Agriculture, Al-Azhar University.

*** Biological and Geological Sciences Department, Faculty of Education, Ain Shams University.

Abstract

Imidacloprid is an insecticide belongs to the new active group nitroguandine which has outstanding potency and systemic action for crop protection against pests. It is one of the insecticides that cause oxidative stress in cells leading to glutathione deficiency.

The present study aimed to investigate the possible protective role of glutathione 0.55mg/kg body weight against the toxic effect of 1/50 LD50 of imidacloprid insecticide in male Japanese quails (*Coturnix coturnix japonica*). Sixty male quails were divided into 4 groups, the first one served as a control, the second received glutathione only, the third group was treated with imidacloprid and the fourth was administrated both glutathione and imidacloprid conjointly.

Birds were treated orally for either three or six weeks followed a recovery period for 3 weeks. The data obtained revealed a marked increase in serum AST, ALT, ALP, glucose, total lipids, total cholesterol, and creatinine of quails treated with imidacloprid only, whereas variable levels of amelioration were detected in treated groups with glutathione plus imidacloprid, specially in levels of glucose, AST activity and creatinine after 6 weeks of treatment. On the other hand, a highly significant decrease in total proteins, albumin and globulin were found in the birds treated with imidacloprid alone, but these returned to levels close to normal in the quails treated with glutathione plus imidacloprid. Albumin/globulin ratio and uric acid level were not significantly changed in all groups. In general, there was appreciable improvement after the recovery period.

Key words : Glutathione, Imidacloprid, Antioxidants, Biochemical Parameters, Japanese quails.

Introduction

The widespread use of pesticides in agriculture and forestry conservation prompted the need of evaluation of the hazards of such materials to wildlife. Recent reports have emphasized that the probability of exposure exists within the indoor living space, as well as in the agricultural and industrial workplace. Moreover, the indoor use of pesticides may create a different and more direct exposure situation. Owing to the extensive use of these chemicals, they are responsible for numerous cases of poisoning in human and non-target wildlife.

Imidacloprid is a new and potent nitromethylene insecticide with low soil persistence and high insecticidal activity at

very low application rates (Brozni *et al.*, 2008). It is used as a crop and structural pest insecticide, a seed treatment, and a flea-control treatment on cats and dogs (Tomizawa and Casida, 2005). Pesticides, in addition to their intended effects, are sometimes found to affect non-target organisms, including humans (Chantelli-Foti *et al.*, 1993 and Chaudhuri *et al.*, 1999). The mechanism of action of imidacloprid differs not only from that of organophosphorus and carbamate compounds, but also from that of the pyrethroids (Soloway *et al.*, 1978).

Insecticides have been observed to accentuate oxidative stress by generation of free radicals in rat tissues, these free

radicals play an important role in toxicity of pesticides and environmental chemicals, by diminishing the antioxidants or altering oxygen free radicals scavenging enzyme system (Banerjee *et al.*, 1999 and Kamboj *et al.*, 2006).

Glutathione acts as an antioxidant and protective agent against insecticides. Antioxidants play an important role in insecticide toxicity protection especially in the hepatic toxicity and prevent the effect of free radicals on vital cells (Geetanjali *et al.*, 1993 and Sinisa *et al.*, 2008).

Many investigators showed that administration of antioxidants can significantly decrease, to some extent, tissue damage induced by different insecticides (Rutcu *et al.*, 2006; Jalili *et al.*, 2007 and Gülden *et al.*, 2008).

Japanese quails (*Coturnix coturnix japonica*) have been widely used as a laboratory bird model in many areas of biological research (Armbrecht and Dewitt, 1963). Only few studies have investigated the effect of imidacloprid toxicity on Japanese quails. Now, many farms in Egypt raise Japanese quails and chickens. So, we have chosen Japanese quails as a good laboratory bird model to investigate the possible ameliorative effects of glutathione as an antioxidant on the imidacloprid-induced biochemical perturbations in male Japanese quails and to re-evaluate these effects after a recovery period.

Material And Methods

A total of 60 male Japanese quails were taken randomly at the adult age from the poultry research farm of the Faculty of Agriculture, AL-Azhar University. The birds were kept under normal laboratory conditions, fed on standard diet and water *ad libitum*. All birds were starved for 12hrs before treatment, but allowed free excess to water. They were allocated at random into 4 equal groups each of 15 birds. Quails in group (A) were considered as controls. Birds in group (B) were orally administered a dose of 0.55mg/kg body weight of glutathione. Quails in group (C) were treated orally with a dose equals to the 1/50 LD50 of imidacloprid insecticide (LD50 of the

insecticide was determined according to the equation of Behren and Karber, 1953). Quails in group (D) were treated with the same doses of glutathione and imidacloprid conjointly. After 3 weeks of treatment 5 birds were chosen randomly from each group and sacrificed. The same treatments continued for the rest of the birds and after additional three weeks, 5 birds were taken out from each group and sacrificed. The rest of birds were left without any treatment for further 3 weeks as a recovery period after which the last batch of birds was killed to assay the impact of recovery.

Biochemical Analysis :

At the end of each experimental period, individual samples were collected after 18h. of treatment cessation during which the birds were deprived of food. Samples of blood were withdrawn and left to clot in a clean dry centrifuge tubes for each bird, then centrifuged at 3500r.p.m. for ten minutes. A portion of the clear supernatant serum was used immediately for glucose determination according to the enzymatic colorimetric method described by Trinder (1969). The remaining serum was frozen at -20°C for subsequent analysis. Serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities were estimated according to the method described by Reitman and Farnkel (1957). Serum alkaline phosphatase (ALP) was determined by the method of Belfield and Golbderg (1971). Serum contents of total cholesterol and total lipids were assayed according to Allain *et al.* (1974) and Knight *et al.* (1972), respectively. Serum total protein and albumin levels were estimated according to the methods described by Doumas (1975) and Doumas (1971), respectively. Serum globulin was calculated according to Latner (1975).

Serum contents of uric acid and creatinine were estimated according to the methods described by Steven (1970) and Husdan & Rapoport (1968), respectively.

Data analysis:

The obtained results were statistically analyzed by using the student "t"-test according to the method of Snedecor and Cochran (1980).

Results

The data represented in table 1 displayed the effect of treatment with imidacloprid and / or glutathione on enzyme activities which reflect the liver function of Japanese quails.

A highly significant increase ($p < 0.01$) in AST, ALT and ALP was detected in groups treated with imidacloprid alone throughout the experimental periods, as compared with the control group. Combination of the antioxidant glutathione with imidacloprid revealed a highly significant increase ($p < 0.01$) in ALT and ALP activities after 3 and 6 weeks of treatment. A partial amelioration was noted in AST activity in groups treated with glutathione and imidacloprid.

AST, ALT and ALP activities showed insignificant changes in quails treated with glutathione or after the recovery period in comparison with the corresponding control groups throughout the experimental duration.

Table 2 shows the changes in serum total cholesterol, total lipids and glucose levels. The data revealed a highly significant elevation ($p < 0.01$) in these parameters in the groups treated with imidacloprid alone after 3 and 6 weeks. Also, a highly significant increase ($p < 0.01$) was recorded in the same parameters in the quails that received glutathione and imidacloprid for a period of 3 weeks. Obvious amelioration appeared in glucose level and it was feeble [significant elevation ($p < 0.05$)] in total cholesterol of groups treated with glutathione + imidacloprid for 6 weeks.

Data from the same table showed insignificant differences between glutathione-treated groups and the control group

throughout the experimental period. A similar profile appeared in the recovery groups.

Results presented in table 3 show that total serum proteins, albumin globulin levels and A/g ratio recorded insignificant difference in groups administered the antioxidant glutathione either alone or in combination with imidacloprid throughout the experiment as compared with the corresponding control groups. However, treating the birds with imidacloprid alone caused a highly significant decrease ($p < 0.01$) in serum total proteins, albumin and globulin throughout the experimental periods with the exception of the globulin level after 3 weeks which revealed a significant decrease ($p < 0.05$).

Serum uric acid and creatinine levels were determined to study the effect of imidacloprid with or without antioxidant on kidney function test. Data presented in table 4 show that serum uric acid level was not significantly changed in any treated group throughout the total experimental period, as compared with the control group. The creatinine level revealed a highly significant increase ($p < 0.01$) after 3 and 6 weeks of treatment with imidacloprid alone. Also, a highly significant increase ($p < 0.01$) was recorded in the groups treated with imidacloprid in combination with glutathione after 3 weeks of treatment, followed by insignificant changes after 6 weeks and after the recovery period as compared with the corresponding control group.

Furthermore, no significant difference was recorded in any of the parameters under investigation in the groups administered glutathione alone throughout the whole experimental duration.

Table(1): Effect of treatment with imidacloprid and / or glutathione on some serum enzymes activities indicative of liver function of Japanese quails.

Groups	AST(U/L)	ALT(U/L)	ALP(U/L)
After 3 weeks			
Control	38.20 ± 0.37	16.90 ± 0.68	126.40 ± 0.41
Glutathione	37.20 ± 1.24 ^{N.S.}	16.30 ± 0.42 ^{N.S.}	126.20 ± 0.40 ^{N.S.}
Imidacloprid	46.00 ± 0.95**	29.50 ± 0.42**	139.70 ± 0.44**
Imidacloprid + Glutathione	42.20 ± 0.92*	27.20 ± 1.75**	129.80 ± 0.35**
After 6 weeks			
Control	39.20 ± 0.80	17.70 ± 0.42	127.90 ± 0.41
Glutathione	38.20 ± 0.37 ^{N.S.}	16.60 ± 0.42 ^{N.S.}	127.90 ± 0.41 ^{N.S.}
Imidacloprid	46.00 ± 0.95**	28.20 ± 1.12**	136.30 ± 0.32**
Imidacloprid + Glutathione	41.60 ± 1.12 ^{N.S.}	24.70 ± 0.42**	130.60 ± 0.29**
After a 3 week recovery period			
Control	40.80 ± 1.24	18.60 ± 0.39	128.30 ± 0.31
Glutathione	39.20 ± 0.80 ^{N.S.}	18.00 ± 0.51 ^{N.S.}	128.30 ± 0.31 ^{N.S.}
Imidacloprid	42.20 ± 0.92 ^{N.S.}	27.20 ± 1.72**	137.30 ± 0.19**
Imidacloprid + Glutathione	39.20 ± 0.80 ^{N.S.}	22.70 ± 0.42**	129.10 ± 0.29 ^{N.S.}

N.S. (Insignificant) in comparison with corresponding control.

* Significant (P < 0.05) in comparison with corresponding control.

** Highly Significant (P < 0.01) in comparison with corresponding control.

Table(2): Effect of treatment with imidacloprid and / or glutathione on serum total cholesterol, total lipids and glucose levels of Japanese quails.

Groups	Serum total cholesterol (mg/dL)	Serum total lipids (mg/dL)	Serum glucose level (mg/dL)
After 3 weeks			
Control	112.80 ± 0.34	202.00 ± 3.74	121.60 ± 1.97
Glutathione	112.80 ± 0.34 ^{N.S.}	202.00 ± 3.74 ^{N.S.}	121.60 ± 1.97 ^{N.S.}
Imidacloprid	140.10 ± 2.23**	272.00 ± 3.74**	165.40 ± 5.79**
Imidacloprid + Glutathione	132.70 ± 2.33**	270.00 ± 2.73**	141.90 ± 2.18**
After 6 weeks			
Control	115.80 ± 0.22	222.80 ± 2.80	124.40 ± 3.74
Glutathione	114.60 ± 2.21 ^{N.S.}	224.60 ± 2.25 ^{N.S.}	124.40 ± 3.74 ^{N.S.}
Imidacloprid	141.80 ± 2.23**	279.00 ± 1.87**	161.50 ± 1.94**
Imidacloprid + Glutathione	129.40 ± 3.43*	260.80 ± 3.89**	135.90 ± 5.35 ^{N.S.}
After a 3 week recovery period			
Control	115.80 ± 0.19	217.80 ± 2.52	125.50 ± 0.56
Glutathione	115.80 ± 0.19 ^{N.S.}	222.60 ± 1.19 ^{N.S.}	127.70 ± 0.86 ^{N.S.}
Imidacloprid	135.80 ± 3.37**	237.80 ± 2.52**	158.10 ± 1.09**
Imidacloprid + Glutathione	128.90 ± 3.26**	237.80 ± 2.52**	132.10 ± 3.07 ^{N.S.}

N.S. (Insignificant) in comparison with corresponding control.

* Significant (P < 0.05) in comparison with corresponding control.

** Highly Significant (P < 0.01) in comparison with corresponding control.

Table(3): Effect of treatment with imidacloprid and / or glutathione on serum total proteins , albumin , globulin and A/g ratio of Japanese quails.

Groups	Serum total protein (g/dL)	Serum albumin (g/dL)	Serum globulin (g/dL)	A/g ratio
After 3 weeks				
Control	5.50 ± 0.13	4.10 ± 0.09	1.40 ± 0.15	3.96 ± 0.29
Glutathione	5.26 ± 0.35 ^{N.S.}	4.10 ± 0.05 ^{N.S.}	1.16 ± 0.36 ^{N.S.}	5.80 ± 1.90 ^{N.S.}
Imidacloprid	4.20 ± 0.07**	3.30 ± 0.08**	0.90 ± 0.15*	4.20 ± 0.89 ^{N.S.}
Imidacloprid + Glutathione	5.10 ± 0.27 ^{N.S.}	3.90 ± 0.04 ^{N.S.}	1.20 ± 0.32 ^{N.S.}	3.30 ± 0.89 ^{N.S.}
After 6 weeks				
Control	5.70 ± 0.09	4.10 ± 0.06	1.74 ± 0.09	2.50 ± 0.16
Glutathione	5.80 ± 0.09 ^{N.S.}	4.10 ± 0.06 ^{N.S.}	1.80 ± 0.14 ^{N.S.}	2.30 ± 0.19 ^{N.S.}
Imidacloprid	4.40 ± 0.07 **	3.40 ± 0.05 **	0.80 ± 0.13**	5.21 ± 1.47 ^{N.S.}
Imidacloprid + Glutathione	5.50 ± 0.13 ^{N.S.}	4.02 ± 0.29 ^{N.S.}	1.54 ± 0.34 ^{N.S.}	2.94 ± 0.47 ^{N.S.}
After a 3 week recovery period				
Control	5.90 ± 0.24	4.30 ± 0.07	1.64 ± 0.27	2.97 ± 0.51
Glutathione	6.02 ± 0.29 ^{N.S.}	4.22 ± 0.04 ^{N.S.}	1.80 ± 0.23 ^{N.S.}	2.65 ± 0.44 ^{N.S.}
Imidacloprid	4.80 ± 0.09 **	3.80 ± 0.09 **	1.30 ± 0.08 ^{N.S.}	2.76 ± 0.22 ^{N.S.}
Imidacloprid + Glutathione	6.02 ± 0.14 ^{N.S.}	4.20 ± 0.04 ^{N.S.}	1.80 ± 0.18 ^{N.S.}	2.35 ± 0.24 ^{N.S.}

N.S. (Insignificant) in comparison with corresponding control.

* Significant (P < 0.05) in comparison with corresponding control.

** Highly Significant (P < 0.01) in comparison with corresponding control.

Table(4): Effect of treatment with imidacloprid and / or glutathione on serum uric acid and creatinine of Japanese quails.

Groups	Serum uric acid (mg/dL)	Serum creatinine (mg/dL)
After 3 weeks		
Control	10.50 ± 0.31	1.50 ± 0.12
Glutathione	10.50 ± 0.32 ^{N.S.}	1.50 ± 0.03 ^{N.S.}
Imidacloprid	11.50 ± 0.39 ^{N.S.}	2.70 ± 0.07**
Imidacloprid + Glutathione	11.10 ± 0.13 ^{N.S.}	2.20 ± 0.04**
After 6 weeks		
Control	11.10 ± 0.23	1.60 ± 0.14
Glutathione	11.30 ± 0.56 ^{N.S.}	1.52 ± 0.06 ^{N.S.}
Imidacloprid	11.80 ± 0.29 ^{N.S.}	2.20 ± 0.03 **
Imidacloprid + Glutathione	11.30 ± 0.56 ^{N.S.}	2.10 ± 0.19 ^{N.S.}
After a 3 week recovery period		
Control	11.40 ± 0.32	1.60 ± 0.05
Glutathione	11.20 ± 0.16 ^{N.S.}	1.50 ± 0.04 ^{N.S.}
Imidacloprid	11.80 ± 0.21 ^{N.S.}	1.80 ± 0.04 *
Imidacloprid + Glutathione	11.10 ± 0.33 ^{N.S.}	1.90 ± 0.15 ^{N.S.}

N.S. (Insignificant) in comparison with corresponding control.

* Significant (P < 0.05) in comparison with corresponding control.

** Highly Significant (P < 0.01) in comparison with corresponding control.

Discussion

The present study is concerned with the effect of imidacloprid on some biochemical parameters. The role of glutathione (as antidote) was also studied. The combination of antioxidants with insecticides may reduce the toxic effects of insecticides on liver tissue (Samuel, 2005 and Sutcu *et al.*, 2006).

Serum aminotransferases activities are known as toxicity markers in the study of hepatotoxicity caused by chemicals (Govindwar and Dalvi, 1990). An increase in the activities of these enzymes is termed as the early recognition of toxic hepatitis. Results of the present investigation revealed a marked elevation in AST and ALT activities throughout the whole experimental period in groups treated with imidacloprid insecticide alone. Similar results were reported by AL - Shinnawy (1994); Helal *et al.* (1997); Gomes *et al.* (1999); Rutcu *et al.* (2006); Gokcimen *et al.* (2007) and Khan *et al.* (2008). The results revealed also that glutathione used conjointly with imidacloprid achieved limited amelioration ($p < 0.05$) in AST activity after 3 weeks and reached to the normal values after 6 weeks and after the recovery period. This indicated that administration of glutathione as an antioxidant with the insecticide under investigation may significantly decrease the extent of damage induced by the insecticide. The Elevation of the aminotransferases activities in blood has been considered as an indicator of tissue damage. The present study recorded high elevation in alkaline phosphatase (ALP) activity of all quails that received imidacloprid alone or conjointly with glutathione. The elevation in serum ALP may be an evidence of obstructive damage in the liver tissue due to insecticidal exposure (Moss *et al.*, 1987). These observations are in agreement with those reported by Narendar and Balachandran (1990); AL-Shinnawy (1994); Kaur *et al.* (2003); Manjula *et al.* (2006) and Sutcu *et al.* (2006).

A high significant increase in serum total cholesterol level was recorded in the present investigation in quails treated with

imidacloprid alone or in combination with glutathione throughout the duration of the experiment. This elevation in serum total cholesterol might be attributed to mobilization of free fatty acids from adipose tissue causing an increase in the availability of acetyl Co - A and subsequent increase in the synthesis of cholesterol. The results are in agreement with the findings of Hanafy *et al.* (1991), Amer *et al.* (1994) and Helal *et al.* (1997). On the other hand, Chetty *et al.* (1993); Al - Shinnawy (1994) and Ismail (2005) reported a decrease in total cholesterol level in some animals treated with different insecticides.

With regard to the serum total lipids, it was found that administration of imidacloprid alone or in combination with glutathione led to a highly significant ($p < 0.01$) increase throughout the experimental period. This hyperlipaemia might have been caused by hepatic damage and the associated biliary obstruction as a results of the insecticide toxicity (Ogata and Izushi, 1991). These results coincide with those reported by Sadurka and Boguszewski (1993); Zaahkouk *et al.* (1996) and Helal *et al.* (1996, 1997).

The present results showed that quails administered the imidacloprid insecticide exhibited a highly significant ($p < 0.01$) elevation in serum glucose (hyperglycemia) throughout the duration of the experiment. In the group treated with the combination of imidacloprid and glutathione, a highly significant increase was observed after 3 weeks, but the glucose level was close to the normal ranges after 6 weeks. The elevation of glucose level can be explain by stimulation of glycogenolysis and gluconeogenesis by the liver with a temporarily loss of endocrine functions of pancreas leading to hyperglycemia (Lasram *et al.*, 2008).

Furthermore, the necessity for extra energy by the liver in the process of detoxification may be reflected in the disturbance in both glycogenolysis and glycogenesis process which, in turn, was manifested as hyperglycemia. In addition, Anam and Maitra (1995) and Helal *et al.* (1997), reported that the increase in serum

glucose level may be induced by a decrease in endogenous insulin release due to damage of pancreatic tissue. These results are in good agreement with those findings obtained by Hore *et al.* (1997); Kalender *et al.* (2004); Reza *et al.* (2007) and Lasram *et al.* (2008).

The present work indicated a significant decrease in total proteins, albumin and globulin level throughout the experimental period in imidacloprid treated group, whereas insignificant change was recorded in groups treated with imidacloprid in combination with glutathione throughout the duration of experiment. The reduction in these parameters might be due to substantially inhibited protein synthesis by the liver as a result of an alternation in the intracellular protein synthesis mechanisms. These results were in agreement with those reported by Shakoori *et al.* (1988); Begum and Vijayaraghavan (1995) and El-Wardany *et al.* (1996), in certain animal species treated with different types of insecticides. Amer *et al.* (1994) reported that the decrease in total protein might be due to reduction of serum globulin level an assumption supported with the disturbances in the immunoglobulin production.

The present study revealed that the concentration of serum uric acid was not significantly changed in all treated groups throughout the whole experiment duration. This might be due to adaptive ability of quails to reduce the risk effects of imidacloprid insecticide on the kidney and to enhance in the glomerular filtration rate (El-Kashoury, 1999).

Concerning the concentration of creatinine, there was a significant increase in imidacloprid treated group till the end of the experiment, whereas in the groups treated with imidacloprid conjointly with glutathione, a significant increase was observed only after 3 weeks of treatment, but there was no change after 6 weeks or after the recovery period as compared to the control quails. Similar results were found by Ahmed (1994) and Gomaa (1995), in a mytren and nuvacron treated rats. Also, many investigators stated that the increase in serum creatinine in experimental animals was an indication of impaired kidney function due to toxicity by many insecticides (Yosef *et al.*, 2003 and Fouda, 2004).

From the above mentioned results, it was clear that the exposure of Japanese quails to imidacloprid caused many disturbances in the biochemical parameters indicative of liver and kidney functions. The conjoint administration of glutathione with the insecticide resulted in partial amelioration.

Finally, it is recommended that imidacloprid due to its hazardous effect to the non-target species should be used with extreme caution and limited to cases where other insecticides with less toxic impacts fail to achieve desired results. It is well recommended to use antioxidants such as glutathione to minimize the toxic effect of imidacloprid.

References

1. Ahmed E K (1994): "Physiological studies on a metryne herbicides as environment pollution with special regard to residues hazards" Ph.D. Thesis. Fac. Vet Med., Cairo Univ.
2. Allain C C ; Poon L S ; Chan C S G ; Richmond W and Fu P C (1974): Enzymatic determination of total serum cholesterol .Clin.Chem.,20:470.
3. Al-Shinnawy M S A A (1994):Metabolic profile and thyroid function in albino rats treated with an insecticide. Ph. D. Thesis, Fac. Educ.,Ain Shams,Univ.
4. Amer T A ;Badawy; M E ;Ibrahim H A and El-Sawi M R (1994):Effects of curacron toxicity on some liver functions.3-lipid metabolism and metabolic products.J.Union Arab. Biol.,2(A):263-282.
5. Anam K K and Maitra S K (1995): "Impact of quinalphos on blood glucose and acetyl cholinesterase (Ache) activity in brain and pancreas in roseringed parakeet (*Psittacula krameri borealis: Newmann*)" Arch.Environ.Contam.Toxicol., 29: 20-23.
6. Armbricht B H and Dewitt J B (1963): "The comparative toxicology of *coturnix* quail with other laboratory animals" 144th Meeting, American chemical society, Los Angeles California, March 31- April 5.
7. Banerjee B D V ; Bhattacharya A ;Pasha S T and Chakraborty A K (1999): Biochemical effects of some pesticides on lipid peroxidation and free radical scavengers. Toxicol. Lett., 107: 33-47.
8. Begum G and Vijayaraghavan S (1995): "In vivo toxicity of dimethoate on proteins and transaminases in the liver tissue of fresh water fish *Clarias batrachus* " Bull. Environ. Contam. Toxicol., 54: 370-375.

9. Behren W and Karber G (1953): "Determination of LD50" Arch. Exp. Path. Pharm., 2: 177-182.
10. Belfield A and Golbderg D M (1971) : Colorimetric determination of alkaline phosphatase (ALP) activity Enzymes In : J.Clin.Chem.Clin.Biochem.,8:561.
11. Brozni D; Marini J ; Tota M ; Jure G and Milin E (2008) : Kinetic evaluation of imidacloprid degradation in mice organs treated with olive oil polyphenols extract . Croatica Chemica Acta , 81 (1) :203 – 209 .
12. Chantelli – Forti G ; Paolini M and Hrelia P (1993) :Multiple and Point procedure to evaluate risk from pesticides . Environ .Health Perspect .101:15 - 20 .
13. Chaudhuri K ; Selvaraj S and Pal A K (1999) : Studies on the genotoxicity of endosulfan in bacterial systems . Mutat . Ras . 439 : 63 – 67 .
14. Chetty K N ; Walker J ; Browen K and Ivie G W (1993): The effects of dietary calcium and chlordecone on cholesterol in serum of rat. Arch. Environ. Contam. Toxicol.,24:365-367.
15. Dumas B T (1975): "Colorimetric determination of total protein in serum or plasma" Clin. Chem.,21 (8): 1159-1166.
16. Dumas B T ; Watson W A and Biggs H G (1971):"Albumin standards and measurements of serum albumin with bromocresol green" Clin. Chem., Acta. ,31: 87-96.
17. EL-Kashoury I A (1999): "Subchronic toxicity studies of imidacloprid, profenofos and carbosulfan and their mixtures on albino rats" Ph . D. Thesis Fac. Of Agric . Cairo, Univ.
18. EL-Wardany I E ; Khalil F A , and Abdalla E B (1996): "Effect of organophosphorus compounds on some physiological responses in laying hens" Food Borne contamination Egyptians Health, University of Mansoura, 8. 26-27.
19. Fouda FM(2004): Haematological and biological and chemical pesticides on the Nile catfish , *clarias gariepinus*. J. Egypt. Ger. Soc. Zool., 43(A): 77-97.
20. Geetanjali D ; Rita P and Reddy P (1993):Effect of ascorbic acid in the detoxification of the insecticide dimethoate in the bone marrow erythrocytes of mice . Food and Chemical Toxicology . 31 (6) : 435 – 437 .
21. Gokcimen A ; Gulle K ; Demirin H ; Bayram D ;Kocak A and Attuntas I (2007): Effects of diazinon at different doses on rat liver and pancreas tissues. Pesticide Biochemistry and Physiology volume 87 (2) : 103 – 108 .
22. Gomaa G M A (1995):"Protective effect of phospholipids and some vitamins against insecticide intoxication in male rats"Ph.D, Thesis , Dep. Zool .Fac.Sci.,Ain Shams Univ.
23. Gomes J ; Daodu A H ; Lloyd O ; Revitt D M and Anil S V (1999): "Hepatic injury and disturbed amino acid metabolism in mice following prolonged exposure to organophosphorus pesticide. Human and Experimental Toxicol., 18:1-33-37-44"
24. Govindwar, S P and Dalvi R R (1990):Age dependent Toxicity of a corn extract in young and old male rats.Vet.Hum.Toxicol;32:23-6.
25. Glden,Z ;Omutag A T ;Ahmet O S and Gksel S (2008):melatonin protects against endosulfan-induced oxidative tissue damage in rats. Journal of Pineal Research, volume 44,(4): 432-438.
26. Hanafy M S M ;Arbid M S. and Afify,M.M.H.(1991):Biochemical and histopathological effects of the organo phosphorus insecticide(Tamaron)in rats. Indian J.Anim.Sci.61(1):43-47.
27. Helal E G E ; Zaahkouk M A and Abdel-Hamid B (1996): "Toxic effect of 3-methylpyridyl carbamate ethiodide on biochemical and hematological aspects of albino rats" AL-Azhar Bull . Sci. Vol. 7(2): 1-18.
28. Helal E G E ;Zaahkouk M A and Hassan A B R (1997):Biochemical and hematological effects of 8- quinaldine dimethyl carbamate methiodide on albino rats. J. Egypt. Ger. Soc. Zool. ,24(A):119-133.
29. Hore S K ;Maiti S K ;Chauhan H V S ;Neelu – Gupta R C ;Koley K M and Gupta N (1997): Effect of long term exposure of mancozeb on clinic bamato biochemical and pathological changes in rats Indian Vet. J.,74(1):26-28.
30. Husdan H and Rapoport A (1968) : Estimation of creatinine by the jaffe reaction. A Comparison methods . Clin . Chem . 14 (3) : 222-238.
31. Ismail D K N (2005):Physiological and histological studies on *Columba livia domestica* treated with an insecticide. M. Sc. Thesis. Faculty of Education , Ain Shams University.
32. Jalili M ; Khanipour R ;Heydari A A ;Farshid F and Salehi S (2007):The effects of vitamin E on Endosulfan- Induced Oxidative stress in Rat Heart . Pakistan Journal of Nutrition 6(4):375-380.
34. Kalender Y ;Kalender S ; Uzunhisar M O ; Ogutcu A ; Acikgoz F and Durak D.

- (2004): Effects of endosulfan on B cells of Langerhans islets in rat pancreas . Toxicology volume 200 ,2 - 3 : 205 – 211 .
35. Kamboj A;Kiran, R and Sandhir R (2006):Carbofuran-induced neurochemical and neurobehavioral alterations in rats: attenuation by N-acetylcysteine. Exp. Brain Res., 170:367-375.
 36. Kaur N ; Srivastava A K ; Bal M S and Kaur H (2003):Subacute oral toxicity of chlorpyrifos in buffalo calves *Bubalus bubalis*. Ind. J. of Vet. Res., 12(1):34-38.
 37. Khan D A ;Bhatti M M;Khan F A ;Naqvi S T and Karam A (2008):Adverse effects of Pesticides residues on biochemical markers in Pakistani Tobacco farmers. Int. J. Clin. Exp.Med.,1(3):274-282.
 38. Knight J A ;Anderson S and Rawie J M (1972):Chemical basis of the sulfo phosphorvanillin reaction for estimating total serum Lipid.Clin.Chem.,18:199-202.
 39. Lanter A L (1975):Clinical Biochemistry, W. B.Saunders Company,Philadelphia,7th edition, P.147-159.
 40. Lasram M M ; Annabi A B ; Rezaq R ; ELj N ; Slimen S ; Kamoun A ; EL-Fazaa, S and Gharbi N (2008): Effect of short-term malathion administration on glucose homeostasis in Wistar rat. Pesticide Biochemistry and Physiology volume 92,3 : 114-119-
 41. Manjula S D ; Benjamin S and Bairy K L (2006):Modulatory effect of vit. C on genotoxic effect of Endosulfan in developing Albino Rats. Drug Research, (5): 113-116.
 42. Moss D W ; Henderson A R and Kachnar J F (1987):Enzymes. -In: Fundamentals of clinical chemistry,3rd Ed.N.W.tietz ed.W.B.Aunders Company , Philadelphia, London.
 43. Narendar S S and Balachandran B (1990):Effect of dimethoate on Wister rats. J. Ecobiol. 2 (4) : 291 – 297.
 44. Ogata M and Izushi F (1991):Effect of chlordane on parameters of liver and muscle toxicity in man and experimental animals.Toxicol,Letters,65(3):327-337.
 45. Reitman S and Frankel S (1957):A colorimetric method for determination of transaminases. Am.J.Clin.Path.,28: 57-63.
 46. Rezaq R ;Mornaqui B ;Kamoun A ;EL-Fazaa S and Gharbi N (2007):Effect of Subchronic exposure to malathion on metabolic parameters in the rat. C. R. Biol. 330(2): 143-147 .
 47. Rutcu R ; Altuntas I ; Yildirim B ; Karahan N ;Demirin H and Delibas N (2006): the effects of Subchronic methidathion Toxicity on rat liver: Role of antioxidant vitamins C and E. Cell Biology and Toxicology, volume 22:221-227.
 48. Sadurska B ; and Boguszewski B (1993): Changes in lipoprotein lipase activity and lipase liver lipids in thiram intoxication. Acta. Biochim. Pol.,40(4):563-567.
 49. Samuel P L (2005): Antioxidants as potentially safe antidotes for organophosphorous poisoning . Current Enzyme Inhibition , volume 1,2: 147-156 .
 50. Shakoori A R ;Ali S S and Saleem M A (1988):Effect of six months feeding of cypermethrin on the blood and liver of albino rats .J.Biochem .Toxicol.,3:59-72.
 51. Sinisa F D ; Gordana C ; Jelena D and Vukosava D (2008):The influence . of vitamin C supplementation on the oxidative status of rat interscapular brown adipose tissue . Journal of Thermal Biology, volume 33,(4) :238-243.
 52. Snedecor G W and Cochran W G (1980) : Statistical methods . Oxford and J. 13 . H. Publishing Co.,7th Ed.
 53. Soloway S B ; Henry A C ; Kollmeyer W D ; Padget W M ; Powell J E ; Roman S A ; Tieman C H ; Corey R A and Horrie C A (1978): Nitromethylene insecticides , In Advances in Pesticide science , Part 2, H. Geissbuhler , G.T. Brooks and P.C.Kearney (Eds.,Oxford, Pergamon Press , 206-217).
 54. Steven S (1970):"Chemistry for medical technologists". 3rd Ed. C.V.Mosby Company , Saint. Louis , USA , P (662).
 55. Sutcu R ; Aetuntas I ; Yildirim, B ; Karahan N ; Demirin H and Delibas N (2006): The effects of subchronic methidathion toxicity on rat liver: Role of antioxidant vitamin C and E . Cell Biology and Toxicology , volume 22, 3: 221-227 .
 56. Tomizawa M and Casid J E (2005) : Neonicotinoid insecticide toxicology: Mechanisms of selective action . Annual Review of Pharmacology and Toxicology vol . 45 : 247-268 .
 57. Trinder P (1969):Determination of glucose in blood using glucose oxidase with an alternative acceptor. Ann. Clin. Biochem., 6:24-27.
 58. Yosef m I; EL-demerdash F M; Kamel K I and AL -Salheen K S (2003): changes in some haematological and biochemical indices of rabbits induced by isoflavones and cypermethrin. J. Toxicol ., 189 (3): 223-234.
 59. Zaahkouk S A Helal, E G E and Hassan A B (1996):Changes in some hematological and biochemical parameters of adult male rats,inresponse to8-hydroxy quinaldine N,N-dimethyl Al-Azhar, Bull. Sci., 7(2): 1401-1410.

التأثير المحسن للجلوتاثيون ضد سمية الإميذاكلوبرايد في السمان الياباني

ایمان جمال الدين عزت هلال* ، سمير عطية محمد زعقوق* ، نبيل فهمى عبد الحكيم** ،

محمد صلاح عبد الحميد الشناوى*** ، أميرة بدر الدين مهني عبد الغنى*

* قسم علم الحيوان - كلية العلوم - جامعة الأزهر (بنات).

** قسم الانتاج الحيوانى - كلية الزراعة - جامعة الأزهر.

*** قسم العلوم البيولوجية والجيولوجية - كلية التربية - جامعة عين شمس.

يعتبر المبيد الحشرى (إميذاكلوبرايد) واحدا من أحدث المبيدات الموجودة والشائعة الاستخدام حاليا في مصر. ولأن استخدام مثل هذه المبيدات يسبب مشاكل صحية عديدة لما تحدثه من أضرار كثيرة سواء للبيئة أو للكائنات الحية .

وقد أجرى هذا البحث بهدف دراسة بعض المخاطر التي تتعرض لها الطيور ومنها السمان الذى يعد واحدا من أفضل نماذج الطيور التجريبية المنتشرة في مصر ، وذلك بتعريضها إلى جرعة مخففة من المبيد الحشرى إميذاكلوبرايد عن طريق الفم. وفي محاولة لتفادى سمية المبيد على الطائر ، فقد استخدم مادة مضادة للأكسدة (الجلوتاثيون) لمعرفة قدرتها على تقليل سمية المبيد . واشتملت التجربة على عدد 60 من ذكور السمان ، قسمت إلى أربعة مجاميع متساوية (ضابطة - معاملة بالجلوتاثيون - معاملة بجرعة 50/1 من الجرعة نصف المميتة للمبيد الحشرى إميذاكلوبرايد - معاملة بالجلوتاثيون مع المبيد) . وعوملت المجموعات على فترتين 3 و 6 أسابيع ، وتم ذبح خمسة من الطيور من كل مجموعة في نهاية كل فترة ، وتركت بقية الطيور لمدة 3 أسابيع بدون أى معاملة كفترة استشفاء ، وأجريت عليها القياسات الكيموحيوية . وأظهرت النتائج ارتفاعا ملحوظا في محتوى المصل من ALT , ALP , AST والجلوكوز والدهون الكلية والكوليستيرول الكلى والكرياتينين للطيور المعاملة بالإميذاكلوبرايد فقط ، بينما كان هناك بعضا من التحسن للزيادة في تلك القياسات في المجموعات المعاملة بالجلوتاثيون مع المبيد خاصة في مستوى الجلوكوز و AST و الكرياتينين بعد 6 أسابيع من المعاملة و كذلك بعد مرور فترة الاستشفاء . وعلى الجانب الآخر كان هناك انخفاض واضح في البروتين الكلى و الألبومين و الجلوبيولين في المجموعات المعاملة بالمبيد وحده وقد عادت إلى التركيزات الطبيعية في المجموعات المعاملة بالجلوتاثيون مع المبيد ، بينما لم يظهر أى تغير ملحوظ على تركيز حمض اليوريك في جميع فترات التجريب . وقد استنتجنا مما سبق أن استخدام الجلوتاثيون يقلل كثيرا من أخطار سمية المبيد على الكبد والكلى .